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CORRESPONDENCE

A case of facial hyperkeratosis induced by a cosmeceutical containing alpha-hydroxy acid and sunlight overexposure successfully treated using oral acitretin

Chemical peels cause destruction of the epidermis and subsequent repair. Postpeel care is important.

An 18-year-old man presented with progressive erythematous keratotic papules with associated greasy debris bilaterally on the cheeks, postauricular, and submandibular areas, and to a lesser extent the bridge of the nose and forehead, that had been present for the past 4 months (Figure 1). He was otherwise in good health and denied any similar symptoms or hereditary keratotic dermatosis among his relatives.

The patient washed his face twice daily with a mild cleanser and gently removed the hyperkeratotic debris using a gauze scrub; however, it would recur within a few days. Removing the debris revealed skin without bleeding or erosions but with mild itching. The patient had applied an acne cosmeceutical containing 15% alpha-hydroxy acid (AHA) to his face, especially his cheeks, once daily before bedtime for 1–2 months before the gradual onset of facial keratosis. The patient had often played basketball outdoors at midday without applying sunscreen. Treatment with oral doxycycline, topical fusidic acid, adapalene, and urea had not improved his condition.

Laboratory findings were within reference ranges. Bacterial cultures yielded negative results. Pathology examination revealed diffuse thick and loosely laminated and ring form orthokeratotic hyperkeratosis of the stratum corneum and opening of hair canals respectively, focal follicular occlusion, a granular cell layer of reduced thickness, and nonsignificant inflammatory infiltration (Figure 2). Based on the clinicopathologic findings, we made a diagnosis of facial hyperkeratosis as a side effect of improper use of a cosmeceutical containing AHA and sunlight overexposure.

We had considered several differential diagnoses including dermatitis neglecta and Darier disease. Poskitt et al[1] first described

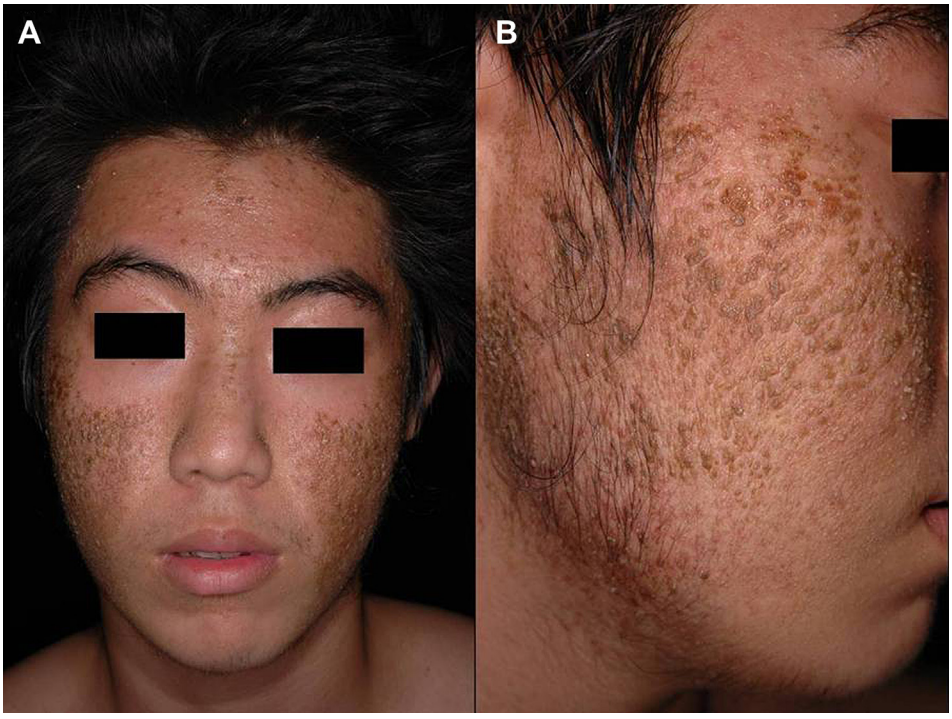

Figure 1 A healthy 18-year-old man with progressive erythematous keratotic papules and associated greasy debris on the cheeks, postauricular and submandibular areas, and, to a lesser extent, the bridge of the nose and forehead over a period of 4 months.

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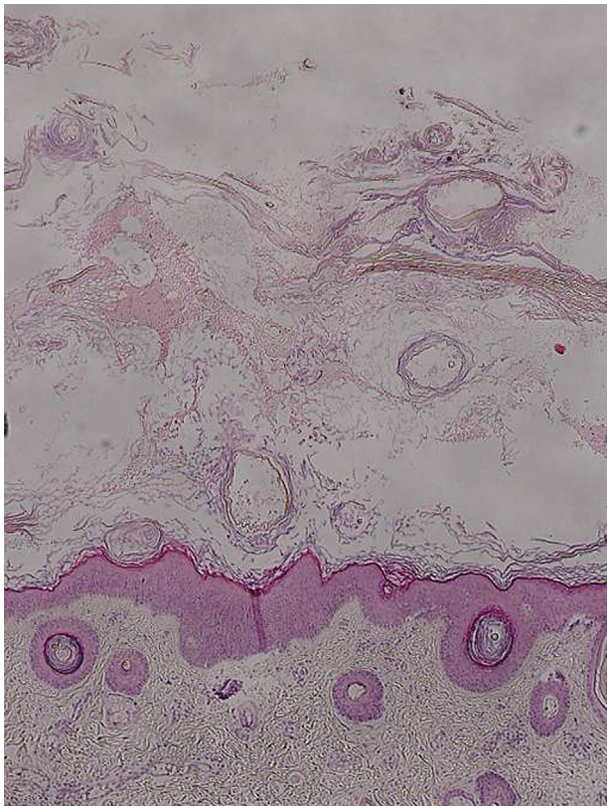

Figure 2 Pathology examination revealed diffuse thick and loosely laminated orthokeratotic hyperkeratosis of the stratum corneum containing ring-form structures from the stratum corneum of the infundibula, focal follicular occlusion, a granular cell layer of reduced thickness, and nonsignificant inflammatory infiltration (H&E stain, 40×).

dermatitis neglecta in 1995. It is associated with the failure to adequately cleanse the skin. An alcohol swab can be used to remove debris, and short-term application of topical keratolytics, such as urea cream, can markedly improve the condition. The symptoms should not recur if good hygiene practice is maintained. Histopathologic features include orthokeratotic hyperkeratosis without substantial cellular infiltration.

Darier disease is a genetic disease of keratinization and presents greasy keratotic papules in seborrheic areas including the face. Histopathologic examination shows suprabasal acantholysis and epidermal dyskeratotic cells.

In our case, we made a diagnosis of facial hyperkeratosis induced by improper use of a cosmeceutical containing AHA and sunlight overexposure based on the following reasons. First, despite the patient cleansing his face on a daily basis, the verrucous patches would recur within a few days of removal. Second, the histologic findings showed no evidence of dyskeratosis. Third, we identified correlation between the use of a cosmeceutical containing AHA, excessive sun exposure, and the onset of facial hyperkeratosis. Finally, the affected areas on the face were consistent with the areas to which the patient applied the AHA cream.[2]

As to normalize the physiologic equilibrium for the dysfunctional epidermis, we treated the patient with 25 mg oral acitretin daily for 2 weeks. After the facial hyperkeratosis had significantly improved, we reduced the acitretin dose to 25 mg/wk to 50 mg/wk for one year, with no evidence of recurrence.

In low concentrations, AHA-containing cosmeceuticals are nonprescription products and are considered safe to use on a daily basis at home. However, unbuffered concentrations as high as 70% can be purchased by physicians for AHA chemical peels. Weekly or biweekly applications of 20–70% unbuffered glycolic acid have been used most often, and the time of application is critical as it must be rinsed off with water after 2–4 minutes. Facial hyperkeratosis is a rare complication and has not been reported to date.[3]

Following ultraviolet radiation exposure, hyperplasia of the dermis, epidermis, and stratum corneum are commonly observed.[4] In 1991, Young et al reported that solar-simulated radiation significantly induced stratum corneum thickening.[5] In 1997, Lock-Andersen et al described that the thickness of the stratum corneum is a determinant of constitutive ultraviolet sensitivity.[6] Sheehan et al later proposed that increased stratum corneum thickening plays a significant role in photoprotection.[7]

In 2004, Lopez et al reported that exposures of two or more minimal erythema doses caused a 10–30% increase in the thickness of the dermis–epidermis layer, and that “diffusion” of the thickening response to neighboring areas occurred in some cases, as far as 4 cm from the exposed region (center-to-center).[2] Waterson et al further described that variation in the stratum corneum or epidermis is a plausible determinant of the biological dose of UV radiation received by the lower epidermis.[8]

In our case, the combination of improper use of a cosmeceutical containing AHA and sunlight overexposure could have enabled ultraviolet radiation to stimulate the exposed mitotically active basal epidermis, resulting in rebound hyperkeratosis as a natural protective response to solar radiation.

In this study, we report the first case of facial hyperkeratosis induced by improper use of a cosmeceutical containing AHA and sunlight overexposure that responded to oral acitretin. Potential risk factors include a higher concentration of free acid, larger volume applied to the skin, facial area, longer duration of contact, higher ultraviolet dose received and inherent genetic keratotic diathesis. It is recommended that AHA concentrations below 10% should be used with strict sun protection measures in susceptible individuals.

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